CHAPTER II.

INFLAMMATIONS OF THE SOFT MEMBRANES OF THE BRAIN; LEPTOMENINGITIS; PURULENT MENINGITIS.

A. Pathological Anatomy. Ætiology.

Inflammations of the soft cerebral meninges occur either at the base or at the convexity of the brain, according as they are primary or secondary—*i. e.*, associated with other diseases—and one can, indeed, with a few exceptions and bearing in mind the transition forms, put it down as a rule that secondary, metastatic meningitis affects the convexity, while a primary meningitis is usually found at the base.

In contradistinction to what takes place in the dura, where the only purulent inflammations that we find are such as have extended by contiguity from neighboring parts, here we have to deal with purulent inflammations alone. This purulent inflammation of the soft membranes of the brain, the leptomeningitis cerebralis, is an infectious disease, and occurs in epidemics as epidemic cerebro-spinal meningitis, or more rarely sporadically, the two forms, however, being ætiologically identical. Besides these, we find developing in the course of tuberculosis, sometimes very early, sometimes late, a specific form of meningitis, the tubercular meningitis.

Pathological Anatomy.—The pathological processes can be traced in the pia as well as in the substance of the brain. In the meshes of the former we find a purulent exudate, which is in rare cases limited to one hemisphere; if it is copious, the pia can easily be stripped from the brain; if it is scanty, this can not be done without loss of substance. The brain substance is œdematous and fills up the skull more than normally, so that the convolutions appear flattened. The ventricles are filled with an unusual amount of fluid (hydrocephalus internus). The hæmorrhages which are recognizable in the brain substance do not exceed in size that of a pin's head, and are either isolated

or are seen especially near the ventricular walls in greater numbers, the so-called capillary apoplexies. Besides these there are other small punctiform hæmorrhages, or rather spots of red softening, and minute hæmorrhages closely grouped together. All these focal changes are to be looked upon as due to the influence of the specific virus. If the process has become a chronic one, then the characteristic features are œdema of the pia, wasting of the brain substance, hydrocephalus internus, and thickening of the ventricular ependyma, which gives to the surface a velvety appearance and changes the shape of the ventricles in a characteristic manner, the normally sharp edges becoming rounded off (chronic meningitis).

In tubercular meningitis we find not only signs of an inflammatory process, but also the formation of tubercles; both, however, do not progress *pari passu*. There may be a very extensive eruption of tubercles and a relatively slight inflammation, or *vice versa*, but always, especially in children, the greater part of the jelly-like exudate is situated at the base (basilar meningitis), between the pons and the anterior perforated space, and imbedded in it are the grayish-white tubercles which are seen as nodules, sometimes as large as millet-seeds, and are found in the greatest numbers among the larger vessels of the fissure of Sylvius, on the chiasma, pons, etc. The vessels are fuller than usual, and small hæmorrhages can occasionally be seen in the pia. The substance of the brain is affected in the manner above mentioned—hydrocephalic effusions into the ventricles are rarely absent, and there is a decided fullness of the choroid plexuses. Foci of softening are noted chiefly about the basal ganglia; they are produced sometimes by the occlusion of an artery, sometimes by the pressure which the exudate exerts on the vessel, or, again, by an arteritis obliterans. Regeneration has been known to occur even in tuberculous meningitis. Dilatation of the ventricles and other signs of an increased intracranial pressure may continue, and collections of fluid in the pia and in the ventricles may still be present, but the fluid may again become clear, the pia moist and non-adherent to the cortex, and the tubercles present no inflammation around them (Wernicke).

A chronic form of basal meningitis, in which the pia is in places either thickened and indurated, or where we have a formation of brittle crusts, may be of a gummatous nature (Wernicke). When a purulent process in the dura extends

into the sinuses we get what is called a thrombo-phlebitis or a (marantic) sinus thrombosis (see Diseases of the Cerebral Veins).

Ætiology.—As has been stated, cerebro-spinal meningitis has to be looked upon as an infectious, sometimes epidemic, disease, the parasitic nature of which was demonstrated by Leyden in 1883. He found in the tissues of the pia and in the turbid cerebro-spinal fluid diplococci, which A. Fraenkel (Deutsche medicinische Wochenschrift, 1886, 13) and G. Hauser (Münchener med. Wochenschr., 1888, 36) recognized as identical with the pneumococcus. Whether or not these cocci gain access to the meninges through the nasal cavities and the foramina of the ethmoidal plate, we are unable to say. Children and young people are more easily affected by the disease than adults, and the infection can be carried by them from place to place. In inclosed and crowded localities, e. g., in prisons and barracks, the disease may become endemic.

But even when there is no epidemic, the disease may appear sporadically anywhere, and then also must be regarded as being just as much of a parasitic nature. Whether the direct influence of the sun's rays is capable of producing meningitis, or at least of favoring its development, has not thus far been sufficiently studied.

A tangible cause for meningitis we find in traumatism of the cranial bones, causing injury to the soft parts, so that the pathogenic organisms can penetrate through the open wounds. The (septic?) *Streptococcus pyogenes* (Eberth), which is less delicate and more resistant than the above-mentioned coccus, has been demonstrated in such cases. If, however, in traumatism, the air remains excluded, as happens in fractures at the base, then the presence of a purulent meningitis is difficult to explain.

The diseases of the bones of the skull, more especially those of the petrous portion of the temporal bone and of the auditory apparatus, play an important part in the ætiology of meningitis. From an otitis media may be developed a caries of the petrous portion of the temporal bone which may perforate the thin roof of the tympanic cavity. The infection extends in such cases along the auditory nerve (Kirchner, Berliner klin. Wochenschr., 1893, 23). Another extension of the inflammation may come from the mastoid cells if an embolus passing from the veins of the bone lodges in one of the venous sinuses, which then becomes the seat of a purulent thrombo-phlebitis.

That the tuberculous meningitis has its origin in tuberculous processes in other organs is clear, and the ætiology is therefore identical with that of tuberculosis in general—i. e., there is invariably an invasion by the tubercle bacillus. It is an interesting fact, however, that though the primary disease in other organs need not necessarily have produced any or at least no marked disturbances, we can still have secondary disease of the pia with the symptoms peculiar to it, which we shall describe. Children especially are not rarely attacked by meningitis, the tuberculous nature of which is only recognized at the autopsy, and we may not have the faintest suspicion of the existence of a previous tuberculous infection. In other cases, however, the meningitis only appears after the pulmonary tuberculosis has made great progress. Caseous bronchial and mesenteric glands, as well as solitary tubercles in the brain, may be the starting point of the meningeal affection, while it less commonly follows tuberculosis of the joints or bones, or tuberculous affections of the intestines and genito-urinary apparatus.

The relation of meningitis to other diseases—i. e., its simultaneous appearance with influenza, pneumonia, scarlet fever, and typhoid fever, ulcerative endocarditis, etc.—has been carefully studied by Huguenin (Correspondenzblatt für Schweizer Aerzte, 1890, 23, 24), but the question whether in those cases we have to deal with a double infection, or whether we have a single noxious agent which produces both the meningitis and the affection which accompanies it, deserves further study. F. Wolff has recently discussed the possible relation of the occurrence of cerebro-spinal meningitis to meteorological conditions—e. g., to the degree of humidity in the atmosphere. The fact that so many cases occur between February and June is perhaps to be regarded as a consequence of the greater humidity which commences in September and does not decrease until April; scarcely any cases occur in July and August, during the period of atmospheric dryness which commences in May (Deutsch. med. Wochenschr., 1888, 38).

It is well established that children and young people are more frequently and more severely attacked by meningitis than older persons, and it seems as if the disease is never found in old age. Early childhood, the period between two and three years of age, furnishes relatively the greatest number of victims and gives the most unfavorable outlook (cf. Kohts, Ueber Paralysen und Pseudoparalysen im Kindesalter nach Influenza, Therapeut. Monatshefte, 1890).

B. Symptoms, Diagnosis, and Treatment.

Symptoms.—The idiopathic, purulent meningitis of the adult usually begins after insignificant prodromal symptoms, such as digestive disturbances, hebetude, etc., with headache, which soon attracts by its severity and its duration the attention of the physician. Exceptionally the patient has hours of comparative ease; usually the headache is so intense that he becomes almost frantic. He tosses about in bed with sighs and groans, and, even when the mind has become dulled, involuntarily again and again puts his hand to his head. Sometimes delirium develops early, to cease again and sooner or later give way to a dull and somnolent condition, which in its turn passes into a deep coma, the immediate forerunner of death.

In some cases the diagnosis is facilitated by characteristic symptoms, such as rigidity of the neck and marked hyperæsthesia of the skin and muscles. The former is especially well recognizable when the patient is asked to sit up in bed, which he can not do without intense pain; the latter is often detected in the examination of the patellar reflexes, which themselves present no particular abnormalities. If we then find besides these symptoms in the beginning of the disease occasional (cerebral) vomiting, a strikingly slow pulse, which is in remarkable contrast with the elevation of temperature (102° and more), and if we carefully examine the pupils, we can not easily make a mistake in the diagnosis. The pupils are usually very much contracted, but may show alternate contraction and dilatation when illuminated for any length of time (Oestreicher, Paradoxe Pupillenreaction, Berl. klin. Wochenschr., 1890, 6). Only exceptionally, however, do we meet with a combination so favorable for the task of the diagnostician. More frequently, as we shall explain at length, he has to encounter considerable difficulties. There is no doubt but that the vomiting is of cerebral origin; but where the center for this is to be sought, whether in the medulla oblongata or, as Hlasko claims (Dorpat, Inaugural Dissertation, 1887), in the corpora quadrigemina, still remains undecided, as also does the question whether or not we are dealing with a functional stimulation of this center. Choked disk and transient paralysis of the ocular muscles are occasionally observed. The former is not easily recognized when the patient quickly passes into sopor; the latter, however, is recognized without difficulty by the strabismus which

it causes and the nystagmus-like movements of the eyeballs. Symptoms of irritation, partly referable to the cortex, in the form of general or unilateral convulsions, muscular unrest, or carphology, partly to individual cranial nerves (grinding of the teeth, trismus, facial spasm), have been repeatedly noted. They seem, however, not always to occur, and for diagnosis must be considered as of minor importance.

The course of purulent meningitis in the adult is different in different cases. As a general rule, however, certain symptoms, especially headache and the rigidity of the neck, sometimes hyperæsthesia of large areas of the skin, persist from the onset and increase, while others, as, for instance, the vomiting and the cranial nerve symptoms, are only transient.

The duration of the disease can be two, three, four, to eight, more rarely ten to fourteen days, and the younger the patient the more dangerous is usually the disease. The patients die, as a rule, without regaining consciousness, but the coma may last for days.

The symptoms of the epidemic contagious (Kohlmann, Berliner klin. Wochenschr., 1883, 17) cerebro-spinal meningitis are on the whole quite similar to those of the idiopathic form. In both the headache is the predominating symptom, and the rigidity of the neck is rarely absent, but in the epidemic more frequently than in the idiopathic form the disease begins with a chill. The course of the fever presents nothing characteristic. It is sometimes of a remittent, sometimes of an intermittent type, the temperature sometimes reaching a height of 104° to 107° F. More or less severe disturbances of consciousness may occur even without a marked elevation of temperature. Among the cranial nerve symptoms, the disturbance in hearing caused by the auditory nerve taking part in the inflammatory process has to be mentioned (Schwabach, Zeitschr. f. klin. Med., 1891, xviii, 3, 4). Visual disturbances are more uncommon, but optic neuritis has been repeatedly noted. If other cerebral symptoms—convulsions, hemiplegia, aphasia—occur, they have to be considered as complications due to an extension of the inflammation to certain parts of the brain substance.

The spinal symptoms, which are superadded, may consist of a distinct tenderness along the whole vertebral column, of a hyperæsthesia of the legs (which is of diagnostic importance), and of twitchings of the extremities. A peculiar, but, as it

appears, extremely uncommon symptom is the so-called flexor contracture of Kernig: the patient when in a sitting posture is unable to extend his knees, because a contracture in the flexors is developed, which disappears as soon as the thigh is no longer flexed at the hip-joint. Bull (cf. lit.) has made some communications on this point. The mechanism of micturition is only influenced when the patient becomes unconscious; then the urine is passed involuntarily. Besides this there are no important bladder symptoms. The urine sometimes contains albumin or sugar, also some tube-casts. Sometimes the quantity voided is greatly increased, a polyuria, which we have to consider as a cerebral symptom.

Other organs rarely take part in the disease. The circulatory, respiratory, and digestive apparatus usually remain normal, and serious stomach affections, endocarditis, and pneumonia, of which we have already made mention above, are seen only rarely as complications. Moderate splenic enlargement often occurs. Among the skin eruptions which sometimes accompany cerebro-spinal meningitis, besides urticaria and (much more rarely) roseola, we have a herpes labialis, which, without being of any prognostic value, possesses a certain diagnostic significance.

The course of epidemic meningitis is still more uncertain and variable than that of the idiopathic form. It may be rapid, and end fatally within a day or a day and a half, in which case convulsions are followed by deep and persistent coma. It may, however, also be protracted, and with remissions, during which the patient is in fairly good condition, may last for weeks. In the beginning of the epidemic usually grave cases are more common, while the longer it lasts the milder they become. It seems as if an attenuation in the virulence of the microörganism had taken place. There occur, besides, abortive cases, in which, while they undoubtedly must be classed with the epidemic disease, only a small, sometimes quite insignificant, part of the symptoms are developed. The period of incubation is from three to five days. Frequently an attack of cerebro-spinal meningitis is followed by certain sequelæ, among the most common of which are headache, pain in the neck, or neuralgias, which may persist for a long time after convalescence.

The symptoms of tuberculous meningitis differ somewhat in children and in adults.

(*a*) In children the disease runs either a very acute or a more chronic course. In the first case only a few days may elapse between the onset and the fatal issue; in the latter, weeks and months may pass before amelioration and recovery, or in these cases also death takes place.

The acute form usually begins suddenly with epileptiform convulsions. Apparently healthy, robust children fall into convulsions and then complain of severe headache and nausea, which is often followed by vomiting; the pulse becomes irregular, and its variations in frequency are more striking than in any other disease. On examination, we find the temperature only moderately elevated, but the patient is very restless, throwing himself about in bed and complaining of pain in the abdomen, chest, etc. Strabismus, trismus, grinding of the teeth, are often noted, and on mechanical stimulation of the skin striking circumscribed red spots, Trousseau's *taches cérébrales*, appear. The patients sigh deeply when examined, or give an unexpected loud, sharp cry, the *cri hydrencéphalique*, a very unfavorable symptom which is of far greater importance than the spots, from the appearance of which we are not justified in drawing either favorable or unfavorable conclusions. The approach of death is announced by an enormous increase in the frequency of the pulse, by renewed convulsions, and deep coma.

The chronic form begins insidiously and gradually, the first thing to attract our attention being the change in the disposition of the child. Previously gay, friendly, playful, and companionable, he becomes peevish, irritable, unmanageable, and willful. On the least provocation he begins to cry and to be naughty, so that the parents find it necessary to punish him. It is not until the sleep begins to be disturbed and the child tosses about all night and groans in its sleep, wakes up in the morning without being rested, and complains of headache, that the parents become apprehensive, and the loss of appetite, the occasional vomiting, the obstinate constipation, and the pale, sickly appearance confirm the fear that a serious malady is on the point of showing itself. The symptoms may for weeks remain obscure; high temperature may alternate with low, a frequent with a slow pulse, without it being possible to say anything definite about the case. Only when one day an epileptiform attack occurs, the headache increases in intensity, the child becomes somnolent, cries out during sleep, shrinks on